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PHYTOPATHOLOGY

VOLUME XIII NUMBER 8

AUGUST, 1923

FURTHER STUDIES ON POTATO SEED TREATMENT

J. C. GILMAN AND I. E. MELHUS[1]

WITH FIVE FIGURES IN THE TEXT

The interpretation of the existing data from experimental plots on potato seed treatment has led to many contradictory statements regarding the value of various seed treatments for controlling seed-borne diseases. This confusion of interpretation of results usually has arisen because an inadequate system of control plots, which, coupled with an insufficient replication of the trials, gave too high an experimental error. In earlier papers the authors[2,3] pointed out three of the variable factors found in such experiments, viz.: the presence after treatment of a greater or less number of viable pathogenes on the seed piece, the inhibitory rather than fungicidal action of certain chemicals used in seed treatment, and the presence of the pathogene in the soil. A system of check plots which will to a marked degree measure these factors was suggested. This system consisted of the use of clean treated, clean not treated and infected not treated seed; the plots were replicated a sufficient number of times to reduce the experimental error to within the limits of practical certainty.

TRIALS OF 1920, 1921, AND 1922

In 1920 and 1921, further data were gathered on the basis suggested above, which confirmed the previous findings; and, in order to increase the value of the observations, the question of the most accurate "measuring stick" in any set of field experiments was studied, use being made of certain experimental plots on the control of black scurf (*Rhizoctina solani*) by seed treatment. In 1920 the amount of disease present on the progeny from the different plots was estimated on the basis of count

[1] Published with the approval of the Director of the Iowa Agricultural Experiment Station.

[2] Melhus, I. E., Gilman, J. C. & Kendrick, J. B.—Fungicidal Action of Formaldehyde. Iowa Agr. Exp. Sta. Res. Bull. 59, 1920.

[3] Melhus, I. E., and Gilman, J. C.—Measuring Certain Variable Factors in Potato Seed Treatment Experiments. Phytopath. **11**: 6–17. 1921.

and weight of the infected tubers from the plots and the percentage of disease thus computed. In 1921 the amount of disease present in the plots was computed both on the basis of stem lesions; that is, the percentage of plants showing stem lesions due to *Rhizoctonia solani*, and on the basis of the presence of sclerotia on the tubers at maturity. The latter was computed by weight. Both years the plots consisted of 200 hills planted in parallel rows. In 1920 each treatment was replicated 5 and the checks 8 times. In 1921 three series of experiments were made. In the series A, the plots were duplicated, the checks triplicated; in series B, the plots were not repeated, and in series C the plots were in triplicate, the checks being replicated 4 times. In the last series 400 hills were planted and then part of them were dug to study the method of reading results by means of stem lesions. The seed treatment studies, particularly, were hot and cold formaldehyde, and mercuric chloride. Copper sulphate, bordeaux dust, and bordeaux mixture were also considered in one or the other of the two years. The data showing the results of these experiments are brought together in the following tables:

TABLE 1—*Results of seed treatment experiments for the control of Rhizoctonia solani in 1920.*

Row	Treatment	Percentage of tubers showing sclerotia	
		By weight	By count
1	Clean, CH_2O 1–240 25° C. 2 hrs.	11.1	10.7
2	Clean not treated	15.2	15.2
3	Infected not treated	24.0	24.7
4	Infected CH_2O 1–240 25° C. 2 hrs.	10.5	14.1
5	Infected CH_2O 1–120 50° C. 2½ min. Covered 2 hrs.	5.3	7.4
6	Infected CH_2O 1–240 6–8° 2 hrs.	17.5	25.1
7	Infected CH_2O 1–240 25° 2 hrs.	4.3	5.3
8	Infected $HgCl_2$ 1–1000 6–8° 1½ hrs.	11.4	11.0
9	Infected $HgCl_2$ 1–1000 25° C. 1½ hrs.	18.5	19.2
10	Infected $CuSO_4$ 1–300 25° C. 2 hr.	21.2	20.1
11	Infected CH_2O 1–120 50° 2½ min. covered 2 hrs.	16.1	16.7
12	Infected CH_2O 1–240 6–8° C. 2 hrs.	28.7	31.8
13	Infected CH_2O 1–240 25° 2 hrs.	16.4	19.9
14	Infected $HgCl_2$ 1–1000 6–8° C. 1½ hrs.	13.3	12.0
15	Infected $HgCl_2$ 1–1000 25° C. 1½ hrs.	18.2	20.3
16	Infected $CuSO_4$ 1–300 25° C. 2 hrs.	11.7	12.5
17	Clean CH_2O 1–240 25° C. 2 hrs.	16.5	15.9
18	Clean not treated	35.0	33.9
19	Infected not treated	17.8	19.3
20	Infected CH_2O 1–240 25° C. 2 hrs.	2.7	3.9
21	Clean CH_2O 1–240 25° C. 2 hrs.	14.8	14.3

22	Clean not treated	25.7	27.6
23	Infected not treated	34.1	38.3
24	Infected CH_2O 1–240 25° C. 2 hrs.	9.6	8.6
25	Infected CH_2O 1–120 50° C. 2½ min. Covered 2 hrs.	12.7	16.8
26	Infected CH_2O 1–240 6–8° C. 2 hrs.	33.0	25.0
27	Infected CH_2O 1–240 25° C. 2 hrs.	12.4	13.7
28	Infected $HgCl_2$ 1–1000 6–8° C. 1½ hrs.	5.0	5.4
29	Infected $HgCl_2$ 1–1000 25° C. 1½ hrs.	6.2	7.2
30	Infected $CuSO_4$ 1–300 25° C. 2 hrs.	17.6	21.2
31	Clean CH_2O 1–240 25° C. 2 hrs.	12.8	15.1
32	Clean not treated	5.5	9.0
33	Infected not treated	47.1	42.5
34	Infected CH_2O 1–240 25° C. 2 hrs.	7.6	9.3
35	Infected CH_2O 1–120 50° C. 2½ min. Covered 2 hrs.	10.7	12.8
36	Infected CH_2O 1–240 6–8° C. 2 hrs.	6.5	8.1
37	Infected CH_2O 1–240 25° C. 2 hrs.	12.9	13.7
38	Infected $HgCl_2$ 1–1000 6–8° C. 1½ hrs.	19.7	21.5
39	Infected $HgCl_2$ 1–1000 25° C. 1½ hrs.	12.5	12.1
40	Infected $CuSO_4$ 1–300 25° C. 2 hrs.	23.0	24.2
41	Clean CH_2O 1–240 25° C. 2 hrs.	17.6	19.2
42	Clean not treated	11.6	11.9
43	Infected not treated	28.6	24.0
44	Infected CH_2O 1–240 25° C. 2 hrs.	14.4	7.9
45	Clean CH_2O 1–240 25° C. 2 hrs.	14.8	14.4
46	Clean not treated	21.8	20.5
47	Infected not treated	43.4	39.3
48	Infected CH_2O 1–240 25° C. 2 hrs.	21.8	21.0
49	Infected not treated B. D.	17.6	18.0
50	Clean not treated B. D.	46.0	38.3
51	Infected CH_2O 1–240 25° C. 2 hrs. B. D.	13.5	13.2
52	Infected CH_2O 1–240 25° C. 2 hrs. B. D.	28.3	24.2
53	Infected CH_2O 1-120 50° C. 2½ min. B. D. Covered 2 hrs.	36.3	31.1
54	Infected $HgCl_2$ 1–1000 25° C. 1½ hrs. B.D.	23.9	20.9
55	Infected $CuSO_4$ 1–300 25° C. 2 hrs. B. D.	11.3	12.6
56	Infected CH_2O 1-240 25° C. 2 hrs. B. D.	22.8	23.7
57	Infected not treated B. D.	47.3	46.3
58	Clean not treated B. D.	26.8	21.4
59	Clean CH_2O 1–240 25° C. 2 hrs. B. D.	20.3	21.0
60	Clean CH_2O 1–240 25° C. 2 hrs.	12.7	12.0
61	Clean not treated	46.0	41.3
62	Infected not treated	36.5	33.9
63	Infected CH_2O 1–240 25° C. 2 hrs.	32.5	32.6
64	Infected Hot HgCl dip	13.3	13.2
65	Infected Hot $CuSO_4$ dip	31.6	34.6
66	Infected Bordeaux paste (10–10–50)	55.5	56.8
67	Infected Bordeaux paste (10–10–50)	38.0	38.8

68	Infected-Paraform dust	37.3	34.7
69	Clean CH_2O 1–240 25° C. 2 hrs.	6.8	9.5
70	Clean not treated	25.3	25.0
71	Infected not treated	47.0	55.6
72	Infected CH_2O 1–240 25° C. 2 hrs.	13.2	17.0
	SUMMARY OF MEANS		
	Clean treated	12.6±0.79	14.04±0.69
	Clean not treated	22.4±2.88	23.92±2.48
	Infected not treated	35.4±2.85	34.16±2.48
	Infected treated	14.1±2.10	14.32±2.05
	CH_2O 1–120 50° C. 2½ min.	11.1±1.2	
	CH_2O 1–240 2 hrs. 25° C.	10.7±1.5	
	CH_2O 1–240 2 hrs. 6–8° C.	22.3±3.4	
	$HgCl_2$ 1–1000 1½ hrs. 25° C.	13.9±1.7	
	$HgCl_2$ 1–1000 1½ hrs. 6–8° C.	12.6±1.6	
	$CuSO_4$ 1–300 2 hrs. 25 ° C.	18.1±1.8	

B. D. The seed was covered with bordeaux dust just previous to planting.

SUMMARY OF TABLE I

The results of 1920 aside from the data concerning the best means of measuring control, showed that the hot formaldehyde treatment (CH_2O 1–120 50° C. for 2 min.) gave practically as good control as the standard formaldehyde treatment, and a little better control than the standard mercuric chloride treatment. The differences in control are of doubtful significance, while the shorter time for the hot method gives it considerable economic value. An interesting point that should be mentioned is the better control with standard formaldehyde at a warm temperature, 25° C. over cold 6–8° C. In the case of the former, the percentage of disease was cut in half. Copper sulphate did not give any better control and the plots showed injury from this chemical. The four check plots maintained the same relative positions as they have in experiments previously reported[1]. Bordeaux paste 10–10–50 and paraform dust were of no control value and very injurious to stand.

In addition to the fungicides mentioned, one set of plots was planted in 1920 in tubers that had been dusted with commercial bordeaux dust just previous to planting and after the tubers were treated and cut. It was thought that the fungicide might act as an antiseptic and prevent the attack of *Rhizoctonia solani*. As will be readily seen from the results, the dusting had little or no effect on the percentage of disease present on the progeny of these tubers, if the percentage of Rhizoctonia on the progeny of any plot is compared with the percentage of Rhizoctonia on

[1] *loc. cit.*

the nearest plot having the same treatment without the dusting. In 6 out of 11 cases the dusted tubers showed more disease present on their progeny than did those not dusted.

These results were estimated in two ways. The progeny from the plots were sorted into those showing sclerotia and those free from sclerotia, and the two groups were weighed and counted, and the percentage of disease calculated from these figures. In the 72 field plots the percentage by count was greater than the percentage by weight in 35 cases. In 28 cases the percentage by weight was greater than the percentage by count and in 9 cases the percentage of both weight and count were equal. The greatest variation was in plot 26 where the

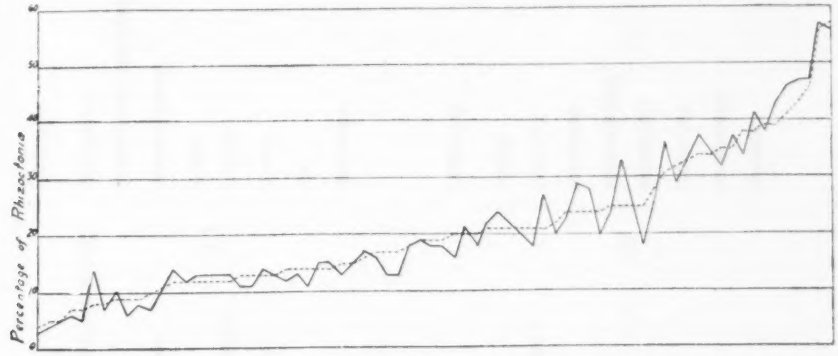

Fig. 1. Comparison of percentage of Rhizoctonia estimated by weight with percentage estimated by count.

Percentage of disease estimated by count.....

Percentage of disease estimated by weight———

percentage by weight was 8 per cent greater than the percentage by count. On the other hand, in the 25 plots showing 25 per cent or more infection the percentage by weight exceeded the count in 16 cases. Figure 1 shows this comparison graphically. An array was made of the percentage values by count and then the percentage value by weight for the same row was plotted. It is apparent that in spite of minor differences the percentage of disease computed by either method is practically the same.

If the mean percentages by count and weight in the four checks which were replicated eight times are compared, we find that in three cases the percentage by count is a little higher than that by weight, but the difference is insignificant. The figures are given in table 1, and shown in histograph form in figure 2. In this figure the solid histographs show the percentage by count; those outlined, percentage by weight.

The means in the checks that were replicated 8 times, show clearly that percentage by weight and count are very close to one another, and therefore it makes little difference whether one uses weight or count as an indicator of degree of control.

It is obvious that the differences are not great and either the count of tubers bearing sclerotia or the weight of the infected progeny may be taken as an indicator of the fungicidal value of a method of seed treatment. From the point of view of mechanical ease and rapidity of performance the measurement by weight is probably preferable.

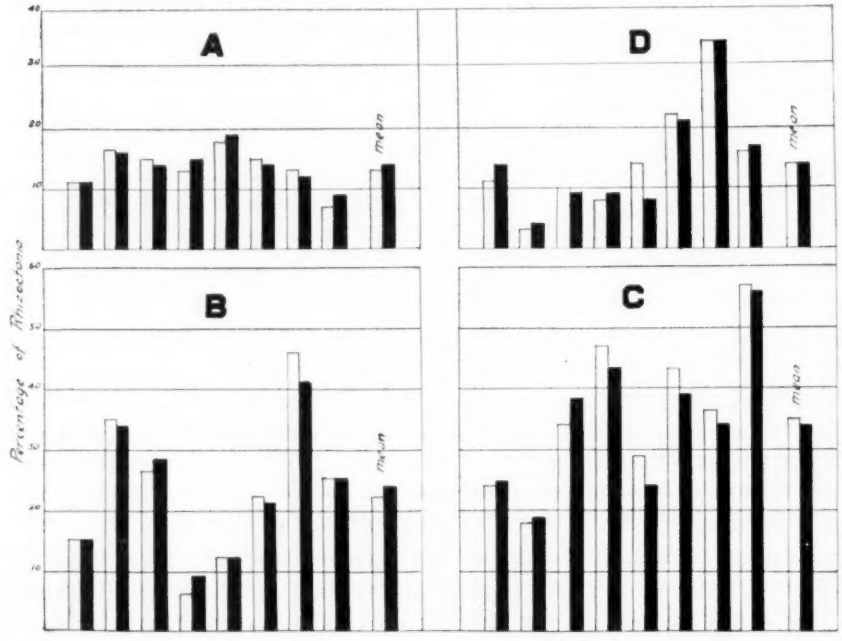

FIG. 2. Comparison of the estimate of Rhizoctonia by count and weight on the four check plots. The solid histographs show percentage of disease by count; the outlined, by weight.

A—Clean treated. B—Clean not treated.
C—Infected not treated. D—Infected treated.

TABLE 2—*Results of seed treatment experiments for the control of Rhizoctonia solani 1921.*

Row	Treatment	Yield of tubers		
		Total wt.	Infected wt.	Per cent infected
		oz.	oz.	
1	Clean treated CH_2O 1–240 25° 2 hrs.	261	67	21.8
2	Clean not treated	378	108	28.5

	3	Infected not treated	284	172	60.5
	4	Infected treated	429	197	45.9
	5	CH_2O 1–120 50° C. 2½ min.	736	146	19.8
	6	CH_2O 1–240 6–8° C. 2 hrs.	700	250	35.7
	7	CH_2O 1–240 25° C. 2 hrs.	514	74	14.4
	8	HgCl 1–1000 6–8° C 1½ hrs.	721	66	9.1
Series	9	$HgCl_2$ 1–1000 25° C. 1½ hrs.	1139	239	20.9
A	10	CaOCl 4 hrs.	678	301	44.3
	11	Clean treated	657	45	6.8
	12	Clean not treated	828	181	21.8
	13	Infected not treated	482	234	48.5
	14	Infected treated	720	224	31.1
	15	CH_2O 1–120 50° C. 2½ min.	628	82	13.0
	16	CH_2O 1–240 6–8° C. 2 hrs.	789	220	27.8
	17	CH_2O 1–240 25° C. 2 hrs.	684	112	16.3
	18	$HgCl_2$ 1–1000 6-8° C. 1½ hrs.	742	168	22.6
	19	$HgCl_2$ 1–1000 25° C. 1½ hrs.	369	5	1.3
	20	CaOCl 4 hrs.	613	95	15.4
	21	Clean treated	608	37	6.09
	22	Clean not treated	482	36	7.4
	23	Infected not treated	368	86	23.3
	24	Infected treated	665	80	12.0
	25	Infected not treated Bordeaux mixture	500	77	15.4
	26	Clean not treated Bordeaux mixture	669	42	6.2
	27	Clean treated Bordeaux mixture	359	41	11.4
	28	Infected treated Bordeaux mixture	535	28	5.2
	29	CH_2O 1–120 50° C. 2½ min. B. M.	679	58	8.5
	30	$HgCl_2$ 1–1000 25° C. 1½ hrs. Bordeaux mixture	582	22	3.7
	31	HgCl 1–1000 6–8° C. 1½ hr. B. M.	463	28	6.0
Series	32	CaOCl 4 hrs. B. M.	708	84	11.9
B	33	CH_2O 1–240 6–8° B. M.	518	84	16.6
	34	CH_2O 1–240 25° C. 2 hrs. B. M.	578	66	11.4
	35	Inf. not treated B. M.	439	93	21.1
	36	Clean not treated B. M.	660	105	15.8
	37	Clean treated B. M.	397	8	2.0
	38	Infected treated B. M.	526	12	2.2
	39	Clean treated	628	19	2.9
	40	Clean not treated	564	28	4.9
	41	Infected not treated	251	0	0
	42	Infected treated	644	56	8.7
	43	Clean treated	434	16	3.6
	44	Clean not treated	434	46	10.5
	45	Infected not treated	262	74	28.2
	46	Infected treated	435	21	4.8
	47	Hot formaldehyde	489	35	7.1
	48	Standard formaldehyde	446	67	15.6

	49 Standard $HgCl_2$	339	66	19.4
	50 Clean treated	378	7	1.8
	51 Clean not treated	288	16	5.5
	52 Infected not treated	466	195	41.8
Series	53 Infected treated	457	40	8.7
C	54 Hot formaldehyde	332	55	16.5
	55 Standard formaldehyde	538	75	13.9
	56 Standard $HgCl_2$	584	55	9.4
	57 Clean treated	466	20	4.2
	58 Clean not treated	602	33	5.4
	59 Infected not treated	524	266	50.7
	60 Infected treated	576	38	6.5
	61 Hot formaldehyde	420	64	13.2
	62 Standard formaldehyde	517	65	12.5
	63 Standard $HgCl_2$	538	46	8.5
	64 Clean treated	466	35	7.5
	65 Clean untreated	330	50	15.4
	66 Infected not treated	391	121	30.9
	67 Infected treated	264	44	16.6

MEANS 1921

Series	Clean treated	9.7 ± 3.0
A	Infected not treated	19.2 ± 3.2
	Infected not treated	43.3 ± 6.1
	Infected treated	27.6 ± 5.5
	CH_2O 50° C. 2½ min.	16.7 ± 1.6
	CH_2O 6–8° C. 2 hrs.	31.5 ± 1.9
	CH_2O 25° C. 2 hrs.	15.5 ± 0.5
	$HgCl_2$ 6–8° C. 1½ hrs.	15.9 ± 3.2
	$HgCl_2$ 25° C. 1½ hrs.	16.1 ± 5.3
	CaOCl 4hrs.	30.6 ± 6.9
Series	Clean treated	4.5 ± 0.7
B	Clean not treated	6.06 ± 0.6
	Infected not treated	13.8 ± 5.7
	Infected treated	10.4 ± 0.8
	Clean treated and B. M.	6.4 ± 2.2
	Clean not treated B. M.	11.0 ± 2.3
	Infected not treated B. M.	18.1 ± 1.3
	Infected treated and B. M.	3.7 ± 0.7
Series	Clean treated	4.4 ± 0.7
	Clean not treated	8.7 ± 1.4
C	Infected not treated	39.9 ± 3.1
	Infected treated	8.2 ± 1.4
	Hot formaldehyde	12.4 ± 1.5
	Standard formaldehyde	13.7 ± 0.5
	Standard $HgCl_2$	11.6 ± 1.9
Summary		
	Clean treated	6.3 ± 1.4
	Clean not treated	12.7 ± 1.9

Infected not treated	37.9 ± 4.3
Infected treated	17.9 ± 3.3
Hot formaldehyde	14.6 ± 1.3
Standard formaldehyde	14.5 ± 0.4
Standard $HgCl_2$	13.8 ± 2.3

SUMMARY OF TABLE 2

The results from the plots in 1921 as shown in table 2 confirmed the data of 1920. The percentage of disease was higher in all cases except checks but the relative positions of the treatments, when they are arranged from the standpoint of control is the same. Hot formaldehyde, standard formaldehyde at 25° C., and standard mercuric chloride all gave practically the same control. The difference between standard formaldehyde treatment at 25° C. and 6–8° C. was again marked. The tubers from the latter plots showed twice as much disease as those from the former.

Bordeaux mixture 8–8–50 was substituted for the bordeaux dust used in 1920, the application being made before the tubers were cut. Otherwise the treatment and comparisons are similar. In this case the bordeaux treated plots show less Rhizoctonia present on the progeny in 10 out of 14 cases. The beneficial effect was insufficient to warrant the treatment, however. The disease was relatively lessened, but the effect of the first treatment was greater than that of the bordeaux mixture. This is clearly shown in the high percentage of disease present when the infected tubers were treated with the bordeaux mixture alone. The greater fungicidal value of the liquid as compared with the dust is probably due to the action of the copper brought into solution on the sclerotia before planting and not to any protective effect in the ground.

During the course of these experiments, the question arose as to whether the percentage of sclerotia on the progeny was an accurate measure of the effectiveness of a given seed treatment, since the injury caused by Rhizoctonia is primarily due to its action on the stem of the growing plant. The deposition of sclerotia on the new tubers might not be a true index of the amount of the fungus that had been present on the seed piece.

TABLE 3—*Comparison of stem and tuber infection with Rhizoctonia solani, 1921.*

Row No.	Treatment	No. Plants	Yields Oz.	Per cent of stem lesions on Rhiz. June 3	Per cent of stem lesions on Rhiz. June 14	Per cent Rhiz. on tubers
1	Clean treated	48	434	0	9	4.2
8	" "	54	378	0	13	3.6
15	" "	56	466	4		1.8
22	" "	56	466	4		7.5

Mean				3.7 ± 0.9		4.5 ± 0.7
2	Clean not treated	52	434	13.4	20	10.5
9	" " "	59	288	13.5	26	5.5
16	" " "	56	602	3.6		5.4
23	" " "	52	330	3.8		15.4
Mean				8.6 ± 1.6		8.7 ± 1.4
3	Infected not treated	46	262	43.4	50	28.2
10	" " "	51	466	50.9	62	41.8
17	" " "	56	524	47.8		50.7
24	" " "	54	391	38.8		30.9
Mean				39.9 ± 3.1		45.4 ± 1.5
4	Infected treated	54	435	11.1	27	16.6
11	" "	55	457	16.1	18	4.8
18	" "	59	576	13.5		8.7
25	" "	54	264	12.9		6.5
Mean				14.2 ± 0.7		8.2 ± 1.45

SUMMARY OF TABLE 3

In the 1921 trials, Series C, each plot was enlarged to 400 sets so as to allow for the digging of part of the sets after the plants were up. The digging was done when the plants were about 4 to 6 inches tall and the number of plants with and without lesions counted. The results obtained are given in table 3 and shown in figure 3. The shaded histographs represent the percentage of the plants dug that had Rhizoctonia lesions; the unshaded columns the percentage by weight of the progeny that showed sclerotia. It is clear that the percentage of plants with lesions and tubers with sclerotia are not always the same, but they have the same general trend and the difference in most cases is but slight. This fact is particularly brought out by the means of the 4 checks in which the differences are less marked than when the individual rows were considered. Stem lesions according to 1921 results, give a similar result to the readings made by means of the deposition of sclerotia on the tubers. The readings obtained by either of these methods probably would not vary materially. It should be stated, however, that the reading on stem lesions should be made early in the life of the plant, for later in the season, as shown in table 3, the fungus spreads from plant to plant and the reliability is thereby lessened, although the relative amount of disease present seems to increase more or less uniformly throughout the plots.

SUMMARY OF TABLE 4

In 1922 the efficiency of hot formaldehyde having been established for Iowa conditions, the efficiency of hot mercuric chloride used alone and in combination with formaldehyde was tested. This combination was made in an attempt to combine the inhibitory action of the mercuric chloride with the fungicidal efficiency of the formaldehyde. The plan of the experiments differed from that used in the preceding trials, in that 8 replications of each plot and 10 replications of each of the 4 check plots were used. This change was made due to the fact that but 100

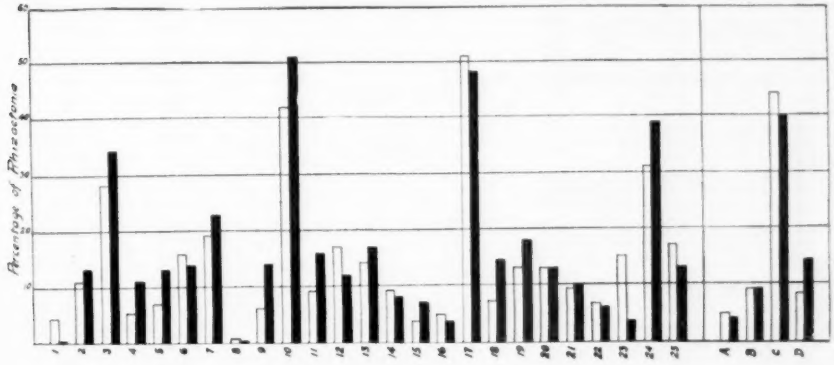

FIG. 3. Comparison of the estimate of the percentage of Rhizoctonia by means of stem lesions (shaded), and the estimate by weight of diseased progeny (outlined).
1, 8, 15, 22—Clean treated.
2, 9, 16, 23—Clean not treated.
3, 10, 17, 24—Infected not treated.
4, 11, 18, 25—Infected treated.
5, 12, 19—Hot formaldehyde.
6, 13, 20—Standard formaldehyde.
7, 14, 21—Standard mercuric chloride.
A—mean of clean treated.
B—mean of clean not treated.
C—mean of infected not treated.
D—mean of infected treated.

sets could be placed in each row. The plots were so arranged, however, that each treatment occupied two adjacent rows which could be combined to constitute a plot comparable to those used in the previous years. In addition since laboratory tests have shown a slight difference between the fungicidal efficiency of mercuric chloride made up in distilled water and that made up in tap (hard) water, plots from potatoes treated in these two solutions were added. The results of this year's experiments are given in table 4.

TABLE 4—*Results of seed treatment experiments for the control of Rhizoctonia solani, 1922.*

Row	Treatment	Yield of tubers		Per cent
		Total wt.	Infected wt.	infected
		oz.	oz.	
1	Clean treated	232	8	3.4
2	" "	356	56	15.7
3	Clean not treated	344	52	15.1
4	" " "	—*	—	— -
5	Infected not treated	—*	—	— -
6	" " "	186	84	45.1
7	Infected treated	508	106	20.8
8	" "	480	86	17.9
9	$HgCl_2$ 1–1000 2 min. 50°	600	468	78.0
10	" " " "	355	199	56.0
11	$HgCl_2$ 1–2000 plus CH_2O 1–240 2 min. 50°	468	304	64.9
12	" " " " " " "	657	236	35.8
13	CH_2O 1–240 2 hrs.	584	168	28.9
14	" " "	446	154	34.5
15	Tap H_2O $HgCl_2$ 1–1000	586	98	16.7
16	" " " "	922	318	34.4
17	Dist. H_2O $HgCl_2$ 1–1000	600	236	39.3
18	" " " "	372	148	39.8
19	Clean treated	609	160	26.2
20	" "	567	176	30.9
21	Clean not treated	720	104	14.4
22	" " "	628	178	28.3
23	Infected not treated	818	162	19.8
24	" " "	462	166	34.9
25	Infected treated	542	184	33.9
26	" "	644	204	31.6
27	$HgCl_2$ 1–1000 2 min. 50°	612	216	34.2
28	" " " "	594	272	45.9
29	$HgCl_2$ 1–2000 & CH_2O 1–240 2 min. 50° C.	750	142	18.9
30	" " " " " "	450	82	18.2
31	CH_2O 1–240 2 hrs.	448	94	20.9
32	" " "	340	50	14.7
33	Tap H_2O $HgCl_2$ 1–1000	312	58	18.5
34	" " " "	350	22	6.2
35	Dist. H_2O $HgCl_2$ 1–1000	562	74	13.1
36	" " " "	492	62	12.6
37	Clean treated	604	18	2.9
38	" "	304	58	19.0
39	Clean not treated	610	105	17.2
40	" " "	420	66	13.3
41	Infected not treated	520	200	38.4
42	" " "	368	128	34.8
43	Infected treated	624	82	13.1
44	" "	631	104	16.4
45	$HgCl_2$ 1–1000 2 min. 50°	506	82	16.2

46	$HgCl_2$ 1–1000 2 min. 50°	315	82	26.0
47	$HgCl_2$ 1–2000 ± CH_2O 1–240 2 min. 50°	418	44	10.5
48	" " " " " " "	382	114	29.8
49	CH_2O 1–240 2 hrs.	534	82	15.3
50	" " "	432	104	24.0
51	Tap H_2O $HgCl_2$ 1–1000	470	100	21.2
52	" " " "	534	114	21.3
53	Dist. H_2O HgCl 1–1000	662	82	12.3
54	" " " "	560	128	22.8
55	Clean treated	584	40	6.8
56	" "	616	122	19.8
57	Clean not treated	666	118	17.7
58	" " "	434	92	21.1
59	Infected not treated	428	120	28.0
60	" " "	400	162	40.5
61	Infected treated	430	60	13.0
62	" "	470	66	14.0
63	$HgCl_2$ 1–1000 2 min. 50° C.	462	142	30.6
64	" " " "	476	71	14.9
65	$HgCl_2$ 1–2000 ± CH_2O 1–240 2 min. 50° C.	644	172	26.7
66	" " " " " " "	542	148	27.3
67	CH_2O 1–240 2 hrs.	430	78	18.1
68	" " "	552	100	18.1
69	Tap H_2O $HgCl_2$ 1–1000	587	20	3.4
70	" " " "	496	120	24.0
71	Dist. H_2O HgCl. –1000	542	70	12.9
72	" " " "	582	110	18.8
73	Clean treated	596	72	12.0
74	" "	546	38	6.9
75	Clean not treated	526	92	17.5
76	" " "	600	92	15.3
77	Infected not treated	604	288	47.6
78	" " "	509	164	32.2
79	Infected treated	440	30	6.7
80	" "	430	32	7.4

Summary

Clean treated	5015	750	14.9 ± 2.3
Clean not treated	4948	899	18.1 ± 0.9
Infected not treated	4295	1474	34.3 ± 1.8
Infected treated	5299	954	18.0 ± 1.9
$HgCl_2$ 1–1000 2 min. 50° C.	3920	1532	39.0 ± 4.8
$HgCl_2$ 1–2000 ± CH_2O 1–240 2 min. 50° C.	4212	1242	29.4 ± 5.0
CH_2O 1–240 2 hrs.	3766	830	22.0 ± 1.5
Tap H_2O $HgCl_2$ 1–1000	4257	850	19.9 ± 2.2
Dist. H_2O $HgCl_2$ 1–1000	4472	910	20.3 ± 2.6

* Rows 5 and 6 were lost at harvest time.

In these plots the amount of diseased progeny in the four check plots remained in approximately the same relation to each other as they held in the three preceding years. The infected treated checks showed approximately the same control as the clean not treated checks. In 1921 the latter were better than the former, while in 1920 the reverse was the case. These facts would indicate that selection of sclerotia-free tubers was approximately as good a control as is seed treatment. The difficulty of securing clean seed, however, makes treatment necessary.

Treatments with mercuric chloride 1–1000 at 50° C. for 2 min., and mercuric chloride 1–1000 plus formaldehyde 1–240 for 2 min. at 50° C. gave no better control than did the untreated checks. In fact, these treatments were of no value for the control of the disease during this season.

Although laboratory experiments as published earlier[1] showed greater fungicidal action of mercuric chloride 1–1000 made up with distilled water, over the same solution made up with tap water (hard water), the field experiments show this difference to be of little practical significance. The tap water solution gave 19.9 ± 2.2 per cent of the progeny diseased, while the distilled water solution gave 20.3 ± 2.6 per cent diseased progeny.

DISCUSSION

The literature shows many different statements as to the relative efficiency of different methods of seed treatment. In 1919, 1920 and 1921 a comparison has been made between the standard cold mercuric chloride and formaldehyde methods and the hot formaldehyde method. Each season each plot consisted of 200 sets replicated from 4 to 6 times. The seed used in these trials each year was as uniformly infected with Rhizoctonia as it was possible to obtain it. The seed in every case was northern seed having been grown either in South Dakota or Minnesota. The percentage of tubers infected with Rhizoctonia sclerotia at harvest time has been used as a "measuring stick."

It is interesting to note the variation in the different plots receiving the same treatment the same year. Note especially the spread in the different plots treated with the hot formaldehyde method in 1919 as shown in figure 4.[2] It varied from 8 to 20 per cent, cold formaldehyde

[1] Gilman, J. C. Effect of hardness of water on the fungicidal value of mercuric chloride solutions (By title) Science n. s. **56**: 262. 1922.

[2] The data for 1919 from which this figure was drawn is given in table IX, Melhus, I. E. and Gilman, J. C. Fungicidal action of formaldehyde. Iowa Agr. Exp. Sta. Res. Bull. 59: 378. 1920.

from 14 to 21, and, mercuric chloride from 6 to 21 per cent. The means in the order mentioned are 15 ± 1.6, 17 ± 1.2, and 14 ± 2.3 respectively, an extreme difference of only 4 per cent. This percentage may well be within the limit of the experimental error. The subsequent two years show similar fluctuations with means differing even less than 4 per cent as in 1919.

In 1920 the mercuric chloride plots showed a variation of 6 to 18 per cent, with a mean of 13.9 ± 1.7; the hot formaldehyde plots, a range of 5 to 16 per cent with a mean of 11.1 ± 1.2, while standard

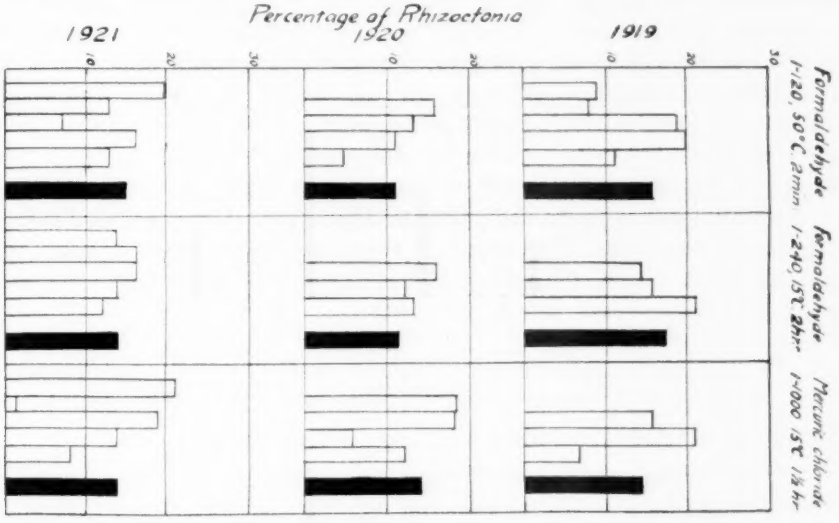

FIG. 4. Comparison of the percentage of Rhizoctonia on progeny from seed treated with hot formaldehyde, standard formaldehyde, and standard mercuric chloride for the years 1919, 1920, and 1921. The shaded histographs show the mean percentage of disease for each series of plots.

formaldehyde showed a range of 4 to 16 per cent with a mean of 10.7 ± 1.5. In 1921 the range for the mercuric chloride plots was 1 to 21 per cent with a mean of 13.8 ± 2.3; that for hot formaldehyde was from 7 to 20 per cent with a mean of 14.6 ± 1.3, and standard formaldehyde varied from 12 to 16 per cent with a mean of 14.5 ± 0.4. The mercuric chloride plots showed the greatest variation about the mean while the formaldehyde plots were more uniform throughout the three years experiments.

The mean of the three years for the three different treatments is practically the same or about 14 per cent. These results are graphically represented in figure 4. They should be compared with the summary of

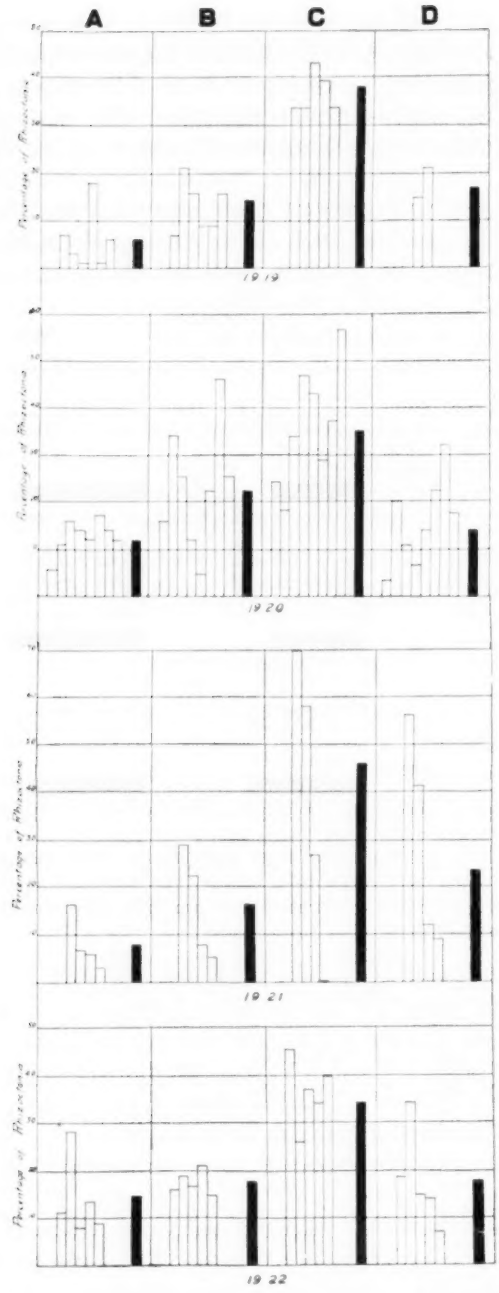

FIG. 5. Comparison of the percentage of Rhizoctonia in the four different check plots over the four years' experiments. The shaded histographs are the means for each series.

A—Clean treated.
B—Clean not treated.
C—Infected not treated.
D—Infected treated.

the check plots figure 5, for the same years, and it will be readily seen that the variability is in most cases less for the different plots than for the check plots. It is this great variability of results which necessitates the replication in order to reach a fair conclusion as to the efficiency of a given treatment.

In the four years' experiments here considered the check plots were replicated 6 times in 1919, 8 times in 1920, 4 times in 1921, and 5 times in 1922. From the data gathered the relative position of the means remains the same, as regards one another, even when but 4 replications were used and in spite of the fact that in 1920 one of the infected not treated plots gave only a trace of Rhizoctonia sclerotia on its progeny. The differing weather conditions, of course, make it impossible to compare the amount of disease except the relative infection resulting in the series of years with the same treatments. From these results four replications seem to be sufficient in the case of the plots used here. Plots of different size or shape might of course require more or might do with less replications but with 200 hills in each plot planted in a row, the plots being parallel, four times seems adequate repetition in ordinary experimental fields. Plots with a fewer number of variable factors such as variety tests or yield, may show significant differences with a less number of replications.

SUMMARY

In potato seed treatment experiments with *Rhizoctonia solani* the efficiency of the treatment can be measured with approximately the same accuracy either by weight or count of the percentage of diseased progeny resulting from that treatment.

Percentage of plants showing stem lesions and percentage of diseased progeny also were of relatively equal value as methods of measuring the efficiency of the fungicides used in seed treatment experiments if the stem lesions were read early in the growing season.

Results obtained over a period of three years showed that hot formaldehyde, cold formaldehyde and mercuric chloride were practically equally efficient from the fungicidal effect produced in controlling *Rhizoctonia solani*. The mean percentages of infected tubers for 1919 were 15 ± 1.6, 17 ± 1.2, and 14 ± 2.3 per cent respectively. For 1920 they were 11.1 ± 1.2, and 10.7 ± 1.5 and 13.9 ± 1.7. For 1921 they were 14.6 ± 1.3, 14.5 ± 0.4 and 13.8 ± 2.3. The results here given show that the formaldehyde experiments gave more constant results than mercuric chloride as evidenced by the greater probable error in the latter plots.

Bordeaux mixture applied as a dust to potato tubers had little fungicidal value as a protection against *Rhizoctonia solani*. Bordeaux mixture 8–8–50 applied as a liquid in which the potatoes were dipped showed a slight fungicidal effect, but one insufficient to warrant its adoption as a practice.

From four years study of seed treatment plots when the plots are 200 hills long in parallel rows, 4 replications will insure fairly reliable results on ordinary experimental fields.

Hot mercuric chloride and a combination of mercuric chloride and formaldehyde showed practically no control of Rhizoctonia in 1922.

BOTANY AND PLANT PATHOLOGY SECTION,
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THE BLACKLEG DISEASE OF THE POTATO. ON THE RELATIONSHIP OF BACILLUS ATROSEPTICUS AND BACILLUS SOLANISAPRUS

SYDNEY G. PAINE AND H. CHAUDHURI

The two organisms *Bacillus atrosepticus* van Hall. and *Bacillus solanisaprus* Harrison produce diseases of potato tubers and stems which, though very similar, bear certain distinguishing features, namely, the effect of the former in producing a melanin reaction in both the tuber and stem which does not appear as an effect of the latter, and the hardening of the vascular bundles by the former but not by the latter; this hardening of the vascular structure causes the "blacklegged" stems to stand erect in the field whereas in the disease described by Harrison (1) caused by *Bacillus solanisaprus* the rotted stems show a dark green, not a black, colour and are so soft that they fall to the ground. Besides these differences between the symptoms which each respectively produces the two organisms exhibit certain cultural differences, notably, in their colony formation, in their power to liquefy gelatine and in gas formation from sugars; *B. atrosepticus* rapidly liquefies gelatine while *B. solanisaprus* is virtually a non-liquefier.

In spite of these differences, however, the experiments of W. J. Morse (2) seemed to show them to be strains of one and the same species; in these experiments, consisting of prolonged cultivation through several transfers in the same medium, the distinguishing characters disappeared and both gave identical reactions. In an earlier communication E. F. Smith (3), describing *Bacillus phytophthorus* Appel, gives his opinion that "*Bacillus solanisaprus* is a closely related, though not identical, organism and the same may be said of *Bacillus atrosepticus*."

Now these organisms have been in the collection of type cultures in the Imperial College laboratory since 1917 during which period they have been "carried on" side by side on the same media and yet have always been quite easily distinguished by their manner of growth. It was therefore considered advisable to repeat the work of Morse and to extend it in other directions in order to settle this question of the relationship of the two species.

The cultures under examination were one of *B. atrosepticus*, in all probability identical with *B. phytophthorus*, isolated by one of us (S.G.P.) in 1917 and one of *B. solanisaprus* obtained in that year from the American Museum of Natural History. Following closely the plan of the experiments of Morse the cultural and physiological characters of these two organisms were compared before and after a series of daily

transfers from tube to tube of nutrient broth. Of the results of these experiments it suffices to say that we were unable to confirm the finding of Morse. With our cultures and in our hands the distinguishing features of the two organisms described above were present equally at the end of this period of attempted "training" as at the beginning of it.

DETERMINATION OF THE THERMAL DEATH POINT

Capillary tubes filled with suspensions of the organisms from an agar slope of 24 hours' growth at 20° C. were heated for ten minutes and the contents plated out and incubated with the following results:—

Temperature	B. atrosepticus	B. solanisaprus
45° C.	20 Colonies	∞ Colonies
47° C.	Nil	∞ Colonies
50° C.	Nil	110 Colonies
54° C.	Nil	3 Colonies

∞ = innumerable.

B. solanisaprus is here seen to be considerably more resistant to heat than *B. atrosepticus*.

INOCULATION EXPERIMENTS

(a) *Inoculation of Potato Stems.* Allowing the organisms to enter the cut end of a stem immersed in a bacterial suspension in water, or prick inoculation of the growing stem gave always consistent differences, namely, a strong melanin reaction and fishy smell with *B. atrosepticus* and a dark green, water-soaked stem and absence of fishy smell with *B. solanisaprus*.

(b) *Surface Inoculation of Sterile Potato Slices.* Both organisms produced very active rotting; *B. atrosepticus* gave its characteristic pink colour and black (melanin) margin to the rotted area accompanied by the fishy smell of trimethylamine, whereas the rot produced by *B. solanisaprus* was a white rot almost devoid of smell. Moreover, the rots in the two cases were of an entirely different chemical order, as shown by the change in hydrogen ion concentration of the rotted tissue. The original Ph. of the potato juice was 6.4 and the final value for the tissue rotted by *B. atrosepticus* was 7.2 as against 4.8 by *B. solanisaprus*.

(3) *Surface Inoculation of Potato Slices at Different Temperatures.* The diameter of the rotted area resulting from equal loop surface inoculations was measured in millimetres after 20 hours' incubation at the temperatures given below:—

	3°	12°	25°	30°	32°
B. atrosepticus	nil	slight rot 20 mm. diam.	active 45 mm. diam.	active 55 mm. diam.	very active 60 mm. diam. slice completely rotted
B. solanisaprus	slight rot 15 mm. diam.	active 50 mm. diam.	complete 60 mm. diam.	complete 60 mm. diam.	complete 60 mm. diam.

The three sets of experiments clearly establish the two organisms as distinct. The authors have been unable to confirm the work of Morse. In searching for an explanation of his results it is conceivable that his cultures were mixed ones obtained from diseased potatoes bearing both species and that by his cultural treatment the one was specially favoured and the other completely eliminated.

IMPERIAL COLLEGE OF SCIENCE AND TECHNOLOGY, LONDON.

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A NEW LEAF SPOT DISEASE OF ONION AND GARLIC

COLIN B. WELLES

WITH PLATE XVII AND ONE FIGURE IN THE TEXT

A severe leaf spot of onion (*Allium cepa* L.) and garlic (*Allium sativum* L.) has been observed since January 17, 1922, at the College of Agriculture, Los Banos, P. I. The disease was first noticed on garlic which on January 31 was so seriously parasitized that it was doubtful if any of the plants would develop to maturity. In the present paper a description is given of the disease and of the causal organism. The writer is publishing these results as a provisional record until the group to which this organism belongs may be satisfactorily classified.

SYMPTOMS

The disease has been found on onion and garlic and on each host the symptoms appear slightly different (Plate XVII A).

On onion: Frequently all of the leaves of a plant are diseased, showing various gradations in seriousness from slight chlorosis to death. Young lesions appear as circular yellow, chlorotic spots, 5 to 3 mm. in diameter. They are most numerous at the outer end of the leaves and gradually decrease in number towards the base. In slightly older attacks the chlorotic spots coalesce at the end of the leaf. Below there are numerous separate lesions giving a mottled appearance.

Later on, the end dries out and turns grayish brown and less frequently the single lesions develop a brownish center made up of dead tissue. In most severe attacks the entire leaf is killed and turns grayish brown.

Fruiting bodies are visible on the older, dead portions of the lesions, or they may appear as black specks on the chlorotic tissue.

On garlic: The distribution and appearance of lesions on garlic leaves are strikingly different from those on the onion. In the first place garlic has flat leaves and the lesions penetrate through the leaves when they are very young. The lesions commence as small, round or irregular chlorotic spots, spreading rapidly until the entire outer end of the leaf is killed. Instead of the appearance of more small lesions below the margin of this lesion the fungus develops and seems to proceed in a mass down the leaf. The diseased tissue is delimited by a narrow band of chlorotic tissue about 3 mm. in width.

After the original lesions fuse, progress of the disease is rapid and it is only a short time until the leaf is killed. Since most leaves show lesions, death of the plants commonly results from infection.

HOSTS PARASITIZED

Inoculations made recently by the writer have shown that Cercospora are very indiscriminate with regard to hosts attacked. It has been found that a species of Cercospora isolated from *Phaseolus lunatus* parasitized all legumes on which it was placed and also parasitized many non-leguminous plants. In view of such varied parasitism, it is impossible to state whether onion and garlic are the only hosts of this organism. However, the organism found on garlic and the one found on onion appear to be the same according to present methods of classification.

ECONOMIC IMPORTANCE

Garlic appears to suffer more from the disease than onion, due, perhaps, to the fact that the garlic plants were more mature and had been exposed to greater possibilities of severe infection.

Out of 198 onion seedlings observed on January 20, four had been killed outright, fifty-four were very seriously diseased, while the remaining 138 had from one to five lesions on each leaf.

A second count of the onion plants on February 6 showed that out of a total of 208 plants observed, 17 were healthy, 117 were slightly diseased, 71 severely diseased, and 3 killed. This shows that the disease was becoming more serious, at least in number of plants attacked. On February 17, from a third count of a total of 186 onion plants, it was found that 2 were healthy, 111 were slightly diseased, 69 were seriously diseased, and 4 were dead. This count, while it presents the actual conditions of the plants on the above date, does not make allowance for dead and fallen leaves which appeared abundantly over the entire patch or for the loss of 22 plants which disappeared after the second count. In each instance the same plants were observed.

On the other hand, garlic suffered much more severely. Out of 230 plants recorded 30 were healthy or so slightly diseased that lesions were not noticeable, 32 plants were slightly diseased, 113 plants were seriously diseased, and 55 killed outright. This count was made on January 17.

On February 2, out of a total of 127 garlic plants, none were healthy, 22 were slightly diseased, 54 were seriously diseased, and 51 entirely dead. From this it appears that 103 out of 230 plants disappeared between January 17 and February 2 or a loss of 44.7 per cent of the plants.

The loss continued even after the plants had been sprayed with Bordeaux mixture on January 24.

Plate XVII B shows the relative size of bulbs obtained from diseased and from healthy plants.

CAUSAL ORGANISM

The organism responsible for the leaf spot disease of onion and garlic belongs to the genus Cercospora and so far as can be ascertained is an undescribed species.

The conidiophores arise in stomata or from injured surfaces of the leaves. They are produced in bunches and in every detail are similar to many of the common Cercospora. The conidiophores are light brown, and measure 47.4 to 165.9 by 5.1 to 7.9 micra with two or three septa when taken from onion, and 50.5 to 168.0 by 5.5 to 8.6 micra with three or four septa when taken from garlic.

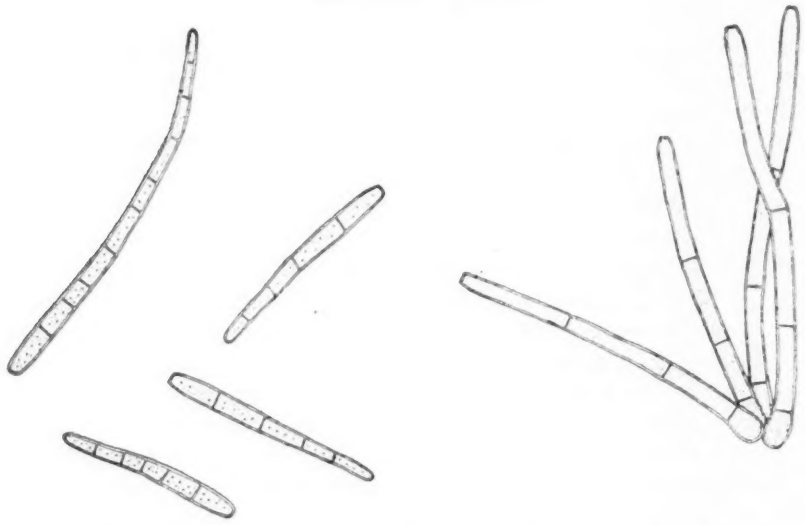


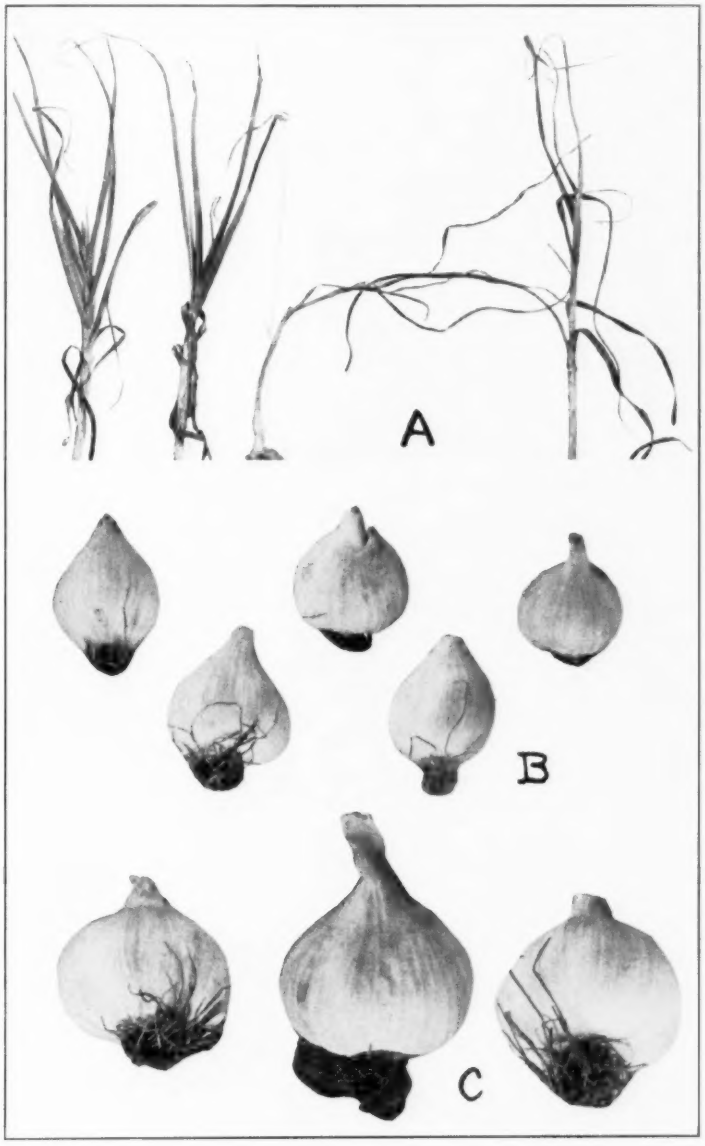
FIG. 1. Conidiophores and conidia of *Cercospora duddiae* n. sp., taken from *Allium cepa*. Drawn with the aid of camera lucida. × 900.

For the most part the conidia are borne terminally on the conidiophores are straight or slightly curved, are moderately short, are thick at the basal end, tapering to a blunt point, and are hyaline. They measure from 48.4 to 79.0 by 6.3 to 7.9 μ with from 3 to 12 septa when taken from onions, and 71.1 to 99.0 by 5.3 to 6.4 μ with from 2 to 15 septa when taken from garlic (Fig. 1).

The name *Cercospora duddiae* is suggested for this organism.

Cercospora Duddiae n. sp.

Spots amphigenous, at first round, later irregular with a border of yellow, chlorotic tissue, having grayish brown centers, 3 to 5 mm. in diameter; conidiophores fasciculate, filiform, simple, frequently genic-



CERCOSPORA DUDDIAE ON ALLIUM SATIVUM

A. Plants of garlic, *Allium sativum*, showing lesions caused by *Cercospora duddiae*. The plants to the left are slightly diseased, those at the right are nearly dead.

B. Bulbs from healthy garlic plants. C. Bulbs from diseased garlic plants. B. and C. same scale.

ulate, 47.4 to 168.0 by 5.1 to 8.6 μ, 2 to 4 septate, light brown; conidia slender, straight or slightly curved tapering from basal end to blunt point, 48.4 to 99.0 by 5.3 to 7.9 μ, 3 to 15 septate, hyaline.

On leaves of onion, (*Allium cepa* L.) and garlic (*Allium sativum* L.) found at Los Banos, Laguna, Philippine Islands.

CONTROLS

The disease on garlic was first observed on January 17 and on January 23 the entire plot of plants was sprayed with Bordeaux mixture. The disease was very serious at that time and little if any benefit resulted from the application.

In view of the fact that the disease is confined to the leaves, the application of Bordeaux mixture as a protective measure should give an efficient control. As the disease has appeared during dry weather, a change in horticultural practice would be of little if any benefit, as Cercospora appear to thrive most luxuriently during periods of heavy precipitation.

SUMMARY

1. A leaf spot of onion (*Allium cepa* L.) and of garlic (*Allium sativum* L.) has been found at Los Banos, P. I. The disease was so serious that the respective crops were reduced approximately fifty per cent.

2. The fungus has been found on onion and garlic although it may be rather omniverous as other Cercospora have been shown to be by inoculation experiments.

3. The causal organism is a Cercospora believed to be an undescribed species.

4. The name *Cercospora duddiae* n. sp. is suggested to designate the fungus.

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NOTES ON THE OCCURRENCE AND GROWTH OF CANKERS OF ENDOTHIA PARASITICA

N. Rex Hunt

During the years 1913–15, the writer conducted a series of spraying tests with lime-sulphur and with Bordeaux mixture as a possible means of controlling chestnut blight. The experimental orchard was located on a shelf near the summit of the Blue Ridge Mountains, fifty miles west of Washington. This shelf slopes gradually down and to the east and is bounded on the west by a short, sharp incline at the top of which is the summit of the ridge and the Virginia-West Virginia state line. The orchard was composed of grafted coppice and native trees mixed. A very few of the grafted trees were Japanese chestnuts, a few were of a superior stock of native chestnuts and the larger part were of the Paragon and related varieties of European chestnuts.

Groups of infected trees were found in the woods to the south and the west of the orchard. Spraying was done with a high pressure power outfit.

A study of the rate of growth of cankers was made (Table 1) and this was used to calculate the probable dates of infection of cankers found (Table 2). The location of cankers was noted (Tables 3, 4) as a possible clue to the source of the spores and to the means of infection. Notwithstanding the enormous number of spores produced by the organism, it seems evident that a relatively low percentage of wounds became infected. Every tree was wounded in nailing on serially numbered brass discs. Countless thousands of wounds were made in cutting and dragging the heavy growth of bush out of the orchard. During the winter of 1914–15 two severe sleet storms broke thousands of twigs and branches. Proximity to diseased trees did not insure quick infection of a tree (Table 5). The amount of growth in large cankers is compared to growth in small cankers and the amount of growth in cankers on grafted trees is compared to growth in ungrafted trees (Table 6). Grafted trees did not blight as readily as ungrafted trees (Table 7). Sprayed trees did not show as much blight as unsprayed trees (Table 8), but spraying was relatively ineffective and not of sufficient benefit to justify the cost.

TABLE 1—*Growth of chestnut blight cankers*

Period of Growth	Winter Sept. 23, 1913 to May 6, 1914	Summer May 6, 1914 to Oct. 23, 1914.	Winter Oct. 23, 1914 to May 14, 1915.	Summer May 14, 1915 to Aug. 26–Sept. 9, 1915.	Fall Aug. 26–Sept. 9 to Nov. 1, 1915.	Fall 1914 to Fall 1915.
No. cankers	3	17	44	30	72	26
Increased diameter of cankers—vertical	1.58 in.	6.06	2.3	4.	1.77	7.8
Monthly rate— "	.22	1.01	.33	1.14	.88	.65
Increased diameter of cankers—horizontal	.5	4.82	1.8	2.85	1.14	5.8
Monthly rate— "	.07	.8	.26	.81	.57	.48

When a new canker was found, it was measured and its probable date of infection calculated on the assumption that it had grown at the average rate during each preceding period of time for which this rate had been determined. For example, if we found a canker with a nine inch vertical diameter on November 1, 1915, we would subtract 8.07 inches, the total average growth (1.77 + 4.0 + 2.3) from October 23, 1914 to November 1, 1915, and assume that the canker had a vertical diameter of 0.93 inches on October 23, 1914. The average rate of growth during the period ending on October 23, 1914 was 1.01 inches per month. $\frac{9.3}{1.01}$ of 30 days = 28 days. Twenty-eight days previous to October 23 would be September 25, 1914. If the similar calculation based on the horizontal diameter threw the infection date back to September 15, the calculated date of infection would be made half way between or September 20, 1914. Many of the cankers were rather small when found and the margin of error was correspondingly smaller.

Winter sleet injuries and increased supplies of spores help to account for the large number of early infections in 1915. The late season infections would not become sufficiently conspicuous to be found during that year.

Numbers in parentheses indicate cankers appearing where tags were nailed on trees. Nearly all of these tags were nailed on the east or on the west sides of the trees. Even without the cankers resulting from

TABLE 2—*Probable infection dates as calulated from the data in table 1.*

	1913	1914	1915
April		1	21
May		6	9
June		5	18
July		10	8
August	1	9	10
September		16	2
October	1	14	1

TABLE 3—*Location of chestnut blight cankers on trees.*

Side of tree on which basal or body cankers came.							
North	N. E.	N. W.	South	S. E.	S. W.	East	West
19	17	18 + (1)*	15	18 + (1)	19 + (1)	33 + (24)	24 + (15)

these wounds it will be seen that there are more cankers on the east and west sides. We are unable to advance a plausible explanation for this.

TABLE 4—*Part of tree infected.*

Year	1911	1912	1913	1914	1915	Total
Location						
Base	1	4	7	15	45	72
Body		2	4	28	42	76
Stub			6	19	20	45
Number			17	22	3	42
Limb	1		3	7	9	20
Branch	1		1	12	27	41
Twig				1	10	11
Top	1	1	2	6	9	19
Root				2	1	3

Base = Infection near ground level;
Body = Below first main divisions of trunk;
Stub = Limb stub on any part of tree;
Number = Point where numbers are nailed on;
Limb = Divisions three inches or more in diameter;
Branch = One-half inch or more in diameter;
Twig = Less than one-half inch in diameter;

The segregation in table 4 is made on the basis of the first or oldest infection on each tree.

TABLE 5—*Infections among trees within ten feet of a diseased tree.*

Date of Infection of diseased trees	No. of trees within 10 ft.	Number of these trees infected each year					No. apparently infected June 30, 1916.
		1912	1913	1914	1915	1916	
1911	6		3	3			0
1912	13			5	2		6
1913	38			9	5	2	22
1914	79				11	2	66

The last examination of the trees was made on June 30, 1916. Relatively few of the 1916 infections would be found in a hasty search made as early as that date.

TABLE 6—*Growth of large cankers vs. small cankers and growth of cankers on grafted vs. ungrafted trees.*

Sept. 1, 1915–Nov. 1, 1915.			Nov. 1, 1915–June 20, 1916.		
No. of cankers	Increase in diameter vertical	horizontal	No. of cankers	Increase in diameter vertical	horizontal
Cankers over 4 in. in vertical diameter					
52	1.64 in.	1.15 in.	68	2.83 in.	2.57 in.
Cankers 4 in. or less in vertical diameter					
21	1.82 in.	1.06 in.	27	2.8 in.	2.33 in.
Growth on grafted trees					
22	1.4 in.	1.2 in.	28	2.61 in.	2.3 in.
Growth on ungrafted trees					
30	1.89 in.	1.2 in.	49	3. in.	2.83 in.

The growth of large cankers as compared to the growth of small cankers was determined for a small number of trees in the fall of 1915. A similar comparison of growth rates was made for cankers found in 1915 and measured on Nov. 1, 1915 and again on June 20, 1916. These data are tabulated in table 6. The amounts of growth made by the large and by the small cankers as shown in this table do not agree but seem to indicate that rate of growth is not greatly affected by the size of the cankers.

A segregation of the same figures was made to compare the relative growth of cankers on native trees with the growth of similar cankers

on grafted trees. Cankers below the graft line of grafted trees were eliminated. These data, as shown in table 6 seem to indicate that cankers grow slightly faster on native trees than on grafted trees.

TABLE 7—*Chestnut blight in grafted and in ungrafted trees.*

	1911	1912	1913	1914	1915	Total No. inf.	Total No. trees	Per cent of trees infected.
Grafted	1	1	3	32	51	88	821	10.27
Ungrafted	1	2	19	26	26	74	306	24.2

Of the total of 1,559 grafted trees, a total of 149 or less than 10 per cent became infected. Of the 829 ungrafted trees, a total of 175 trees or 21 per cent became infected. Data for part of the orchard was segregated by years, showing the erratic proportions given in table 7. Ten grafted trees died from chestnut blight. These trees averaged 5½ inches D. B. H., and died in an average of 2.2 years after the date of infection.

42 ungrafted trees died from the disease. These trees averaged 4 inches D. B. H., and died on an average of 2 years after the date of infection. A few trees were destroyed before notes were taken as to whether or not they were grafted. No distinction was made between cankers above and below the graft line in making these tabulations.

TABLE 8—*Effect of spraying for chestnut blight*

	1911	1912	1913	1914	1915
Number of healthy trees at beginning of year.	2526	2522	2514	2460	2194
Per cent infected during year.	.16	.28	1.6	4.5	7.6
Number of trees sprayed			1550	1493	1424
Per cent of sprayed trees infected during spray season.			1	2.7	4.3
Number of trees unsprayed			964	967	662
Per cent of unsprayed trees infected.			2.3	6.7	7.8

Some trees were cut down in 1914 and 1915. Of the 2,194 trees unblighted at the beginning of the year 1915, 108 were cut down before the end of the season. A number of these felled trees had blighted and all are included in the grand total. They are not included in the totals of sprayed and unsprayed trees. The benefit of spraying was so slight that any protection afforded would seem to be very transitory. Omitting such infections of sprayed trees as were calculated to have occurred before or after the spraying season for each such tree, the results of

spraying as shown by table 8 are still very poor. Extending the length of the spraying season could not be expected to greatly improve results. Trees became infected even when sprayed every fifteen days or when sprayed after each rain. The foliage of lime-sulphur sprayed trees was injured but this injury was not noticeable a few days after spraying. Bordeaux sprayed trees had a characteristic dark blue-green foliage and lost their foliage a week or ten days earlier than lime-sulphur sprayed trees. There seemed to be little choice between the two sprays from either the standpoint of disease control or of effect on the host. Neither spray produced any benefit commensurate with the cost.

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FIELD CONTROL OF TOMATO MOSAIC

MAX W. GARDNER AND JAMES B. KENDRICK[1]

WITH ONE FIGURE IN THE TEXT

The importance of the perennial weed relatives of the tomato as winter carriers of the mosaic disease has been pointed out in our previous publications[2] and the eradication of the perennial ground cherries (*Physalis subglabrata*, *P. virginiana*, and *P. heterophylla*) and horse nettle (*Solanum carolinense*) in and near tomato plant-beds and fields has been recommended as a mosaic control measure in Indiana.

During the summer of 1922 a cooperative trial of these recommendations on a field scale was carried out in cooperation with Mr. Ralph Kemp, a tomato grower and canner. The work was located near one of Mr. Kemp's factories at Kempton, Indiana, in a section of the corn belt very favorable for the production of canning tomatoes and likewise a section where the mosaic disease is annually prevalent to a serious extent, presumably because of the abundance of the weed carrier, smooth ground cherry (*P. subglabrata*).

The tomato plants were grown in a series of 24 plant-beds and two field beds which were located in what was previously a sod pasture in order to obtain as great freedom as possible from the disease-carrying weeds, since these are abundant mainly in cultivated soil. The plants were transplanted to three fairly well separated fields, not far from the plant-beds. Field 2 consisting of five acres and field 3 consisting of 30 acres were planted about the third week in May, and field 1 consisting of ten acres was planted about two weeks later.

A careful search for the mosaic-carrying weeds was made in and around the plant-beds on May 11 and none were discovered. On May 22, however, a number of young *Physalis subglabrata* plants were found in three areas near the plant-beds but none showed mosaic.

Upon this first appearance of the weeds, the area comprising the plant-beds and fields 1 and 2 was surveyed once a week and a vigorous campaign to keep down the weed carriers in and around fields 1 and 2 was begun. No effort was made to keep the weeds out of field 3, which was to serve as a check.

[1] Contribution from the Botanical Department, Purdue University Agricultural Experiment Station, LaFayette, Ind.

[2] Gardner, Max W. and Kendrick, James B. Overwintering of tomato mosaic. Bot. Gaz. **73**: 469–485. 1922.

———Tomato mosaic. Purdue Agr. Exp. Sta. Bull. **261**: 1–24. 1922.

On May 29, *P. subglabrata* was appearing in abundance and about a hundred, of which 12 showed mosaic, were cut down about fields 1 and 2. A few appeared in the plant-beds but did not show mosaic. A week later a still larger number of the weeds had appeared and hundreds of the *P. subglabrata*, including 69 cases of mosaic, were cut out in and around fields 1 and 2, particularly in low areas. One colony of *Solanum carolinense* and later a few *P. heterophylla* plants were also destroyed. At this time it was necessary to clean up only the edges of the fields and the fence rows since the fields were being thoroughly cultivated. An attempt was made to keep the weeds out of a zone 100 feet wide, around the fields. It was found useless to pull the weeds by hand and the hoe was used.

The Physalis eradication work was repeated on June 14, June 20, June 28, and June 29. An abundance of new shoots was found each time and a continual effort was necessary to keep them cut down. In addition it became evident early in June that horse cultivation could not be depended upon to destroy the Physalis between the tomato rows and it was necessary to have men go over the fields with hoes at least once a week. This campaign was continued until July 19 in field 2, and until early August in field 1. Eradication of the Physalis proved extremely difficult because of its great abundance and the ease with which younger shoots escaped detection and also because new shoots were sent up promptly from the rootstocks after the older shoots were cut off. There was a very considerable percentage of mosaic among the weeds.

In the meantime, field 3 received ordinary cultivation and no special effort was made to combat the Physalis which was equally as abundant as in the other fields and likewise showed a considerable percentage of mosaic.

The first mosaic on tomatoes was found on July 5, five weeks after it was noted on Physalis. A careful inspection of field 2 revealed 12 cases of tomato mosaic among the 18,400 plants in the field or 0.06 per cent. These cases were rogued out, but later no attempt was made to rogue the diseased tomatoes. The results of subsequent surveys to ascertain the relative prevalence of mosaic in the two fields (1 and 2) in which an attempt was made to eradicate the weed carriers as compared with the field (3) in which no such effort was made are shown in table 1.

These results show that eradication of the weed carriers exerted a distinctly retarding effect upon the inception and development of the tomato mosaic epiphytotic, since the infection in field 2 was less than

TABLE 1—*Effect of weed eradication on tomato mosaic.*

Date Examined	Field No. 1 Weed carriers eradicated		Field No. 2 Weed carriers eradicated		Field No. 3 Weed carriers not eradicated	
	No. of plants examined	Per cent mosaic	No. of plants examined	Per cent mosaic	No. of plants examined	Per cent mosaic
July 5			18,400	0.06		
July 19	22,105	0.12	18,400	0.4		
Aug. 8	10,800	0.33			5,424	7.0
Aug. 16			6,800	1.0		
Aug. 29	6,400	0.43	3,600	1.8	1,720	14.2

two per cent by the last of August, while in field 3, it was 7 per cent on August 8 and 14 per cent the last of the month. An even more striking control was obtained in field 1, which however, was planted about two weeks later than the other two fields. In this field there was only one-half of one per cent of mosaic the last of August. These results are summarized graphically in figure 1.

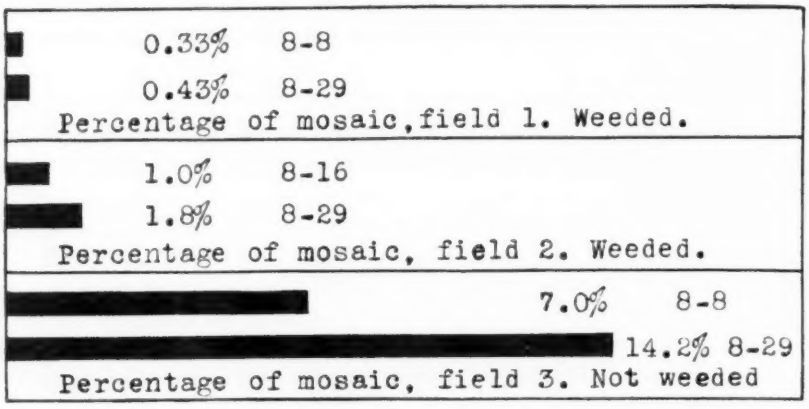

FIG. 1. Tomato mosaic control by suppressing the weed carriers.

Since these fields produced throughout September there was no doubt a general spread of mosaic after this data was obtained, but it is to be remembered that it is the mosaic of early inception which causes the greatest loss.

To summarize this attempt to control mosaic in the field, it may be said that eradication of the mosaic-carrying perennial weeds in and near the plant-beds early in the season and in and around the fields during June and July apparently held tomato mosaic in check. Many difficulties are met with in a weed eradication campaign of this sort, chief of which is the necessity of repeating the process every few days because of the promptness with which new shoots appear. Where the weeds are as abundant as in the tomato regions of central Indiana, many shoots may be overlooked and thus remain a source of infection. It seems entirely feasible for plant growers to destroy these weeds in and near the plant-beds and it is indeed highly desirable to wage an active campaign against them in and around the fields during the early part of the season. In the canning crop it hardly seems advisable to recommend rogueing out the early cases of mosaic tomatoes, and it appears desirable to focus attention for the present upon the eradication of the weed carriers.

A means of total eradication of such weeds is much to be desired. In this connection it is of interest to note that in a field near Frankfort in which *Physalis subglabrata* had occurred plentifully in 1919 when it was cropped to tomatoes, in 1920 when it was cropped to corn, and in 1921 when it was cropped to oats and seeded to clover, very few of the weeds could be found in 1922 when the field produced clover hay. No doubt the rootstocks were still alive, however, but the weeds appeared only in spots devoid of clover such as those marking the location of grain shocks.

As to the persistence of mosaic in the weed carriers, rootstocks of mosaic *P. subglabrata* which were planted in a sunken frame in a garden in 1920 and which sent up mosaic shoots in 1921 as previously reported (l. c.), again sent up mosaic shoots in May, 1922. Furthermore, a rootstock of mosaic *P. heterophylla* planted in 1921 sent up a mosaic shoot in May, 1922. In the field near Frankfort, mentioned above, which had not been in tomatoes since 1919, a few cases of mosaic *P. subglabrata* were found in 1922.

Observations made in 1922 further corroborate our previous observations in regard to the relation of the weed carriers to tomato mosaic (l. c.). The weeds occur abundantly in the fields and are very commonly affected with mosaic. They have also been found commonly in and about plant-beds. *Physalis subglabrata* and *Solanum carolinense* seem to be the principal offenders, the former predominating in the richer soils of central Indiana and the latter in the poorer soils of southern Indiana.

Microscopic examination showed the epidermal cells to be affected. They had become filled with a brown pigmented substance but appeared unharmed in other respects. On the thoroughly whitened pods the stomatal guard cells, which now contained no green matter, were also affected. The cells did not plasmolyse when treated with strong or sugar solutions.

At the same time pods were exposed to the arc light similar pods were likewise exposed, but under the protection of a pane of ordinary window glass. No scald appeared in these pods at any time. At the time the pods were exposed to the sun on March 30 and 31, other pods were similarly exposed, protected by glass, and on them no scald appeared. Ordinary glass is known to be absorptive of light of very short wave length.

The leaves of the plants exposed to the arc light received a similar injury to that of the pods. The epidermal cells of the lower surfaces were killed and colored a deep brown. The cells of the upper epidermis were not so strongly affected. The cells containing chlorophyll were stimulated to increased chlorophyll production and became a deep green. Gradually, however, the leaves began to change color. As the epidermis was killed the leaf slowly lost water. It became unevenly desiccated. The appearance was peculiar in that there were many shades of green in no definite relation with regard to position on the leaf or intensity in color. The leaf was mottled, but not with the definateness of the mottling of bean mosaic. In three weeks' time the leaf had become well dried and assumed a more general uniform color, though it had not fallen from the stem.

The appearance of the artificially scalded leaves was similar to one always observed on a small percentage of bean leaves in the field at about the time when sunscald of the pods appeared. Heretofore there has been no suitable description for it, and no cause could be assigned. It had mostly the appearance of some deviation from mosaic. It is now apparent that the leaves were suffering from a natural scalding which acted very mildly and over a comparatively long period of time.

The stems and petioles were likewise browned, having the exact appearance of scalded stems as found in the field under natural conditions.

DISCUSSION

The burning of plant cells by chemically active light rays, or light of short wave length, has not been much observed in nature. A thick atmosphere, especially when holding a large amount of suspended

water vapor, is so absorptive of light of very short wave length as to practically prevent its reaching the earth. That some light of short wave length does penetrate to the lowest levels is attested to by the fact that in the tropics cataract of the eye due to coagulation of the epidermal layers of eye tissue is frequently encountered among elderly people (4), although certain conditions of health may predispose the eyes to cataract. In this case, however, the effective wave length is apparently above λ 2,650.

The Colorado bean section, at an altitude of 4,700 feet (1,430 meters) and with a relative humidity of less than 20 per cent for a few hours during the heat of the day, would receive a much larger portion of ultra-violet light than those sections at an altitude varying between sea level and 800 feet (244 meters) and with a relative humidity of from 60 to 80 per cent. Brandenburg (3, p. 862) says that the prevailing lack of moisture in Colorado air is favorable to increased intensity of the direct rays of the sun. Solar temperatures obtained from a black-bulb thermometer in vacuo, exposed to the direct rays of the sun at midday, average during the different seasons about 60° F. higher than the air temperature.

The difference in altitude between western and eastern bean-growing districts may make the sunscald definitely apparent only in the West.

The physiology of the action of ultra-violet light on individual plant cells seems not to be thoroughly understood. Maquenne and Demoussy (8) endeavored to determine this point by comparing the phenomenon shown in leaves dipped in water at different high temperatures, in alcohol, ether, and chloroform vapors, and injured mechanically, with that in leaves sprayed with ultra-violet light. They concluded that the brown color is caused by the action of diastase within the cell acting on the starches which have been released upon the death of the cell. Agulhon (1) finds that ultra-violet light destroys not only the cells but also the active products of the cell. The visible light is active on diastase in the presence of oxygen; the ultra-violet light is able to decompose water, form hydrogen peroxid, and then, in the presence of either free oxygen or only hydrogen peroxid, destroy the diastases by the phenomena of oxidization. Stoklasa (10) says that the browning of leaves exposed to ultra-violet light appears after the protoplasm of the epidermal cells is killed by the chemical action of light. Bovie (2) subjected egg white and ox serum to ultra-violet light in a quartz tube. A coagulum was formed which was similar in all respects to a coagulum formed by heat in these same proteins.

CAUSE OF SUNSCALD OF BEANS

H. G. MacMillan

A scalding of the pods and other parts of the bean plant (*Phaesolus vulgaris*) as a result of exposure to the sun was noted by the author (5) in 1918. No constituent of the sun's light was at that time designated as responsible for the scalding. The yearly recurrence of the scald has stimulated further study of this problem. Experiments were made during the winter of 1922 to determine, if possible, what factor or factors caused the scald. Bean plants were grown in the greenhouse and subjected to artificial heat and light.

HEAT

Heat is a property of the sun which might cause the scald. It injures beans. MacMillan and Byars (6) observed injury to beans caused by heat estimated to have reached a temperature of at least 54.4° C. No scalding was observed at the time.

In order to determine the effect of mild heat, the ripening pods of beans were exposed to heat radiated from an electric flatiron. Thermometers were placed beside the pods in such positions as to receive an equal amount of heat. No effect of the heat was observed on the pods until the temperature of the thermometer bulb had reached 55.0° C., and had been maintained for 0.5 hour. The effect was not immediately observed but appeared after 24 hours. The surface was dried, white to creamy white in color, and eventually much shrunken. No brown or discolored areas appeared at any time. The unaffected portion of the pod remained as before and reached maturity in due time. No symptom, however, slight, appeared on the heated pods which at any time resembled the true sunscald.

LIGHT

The harmful effect of light on plants has been noted frequently. Maquenne and Demoussy (7) exposed leaves of ivy and fig to the unprotected light of a mercury vapor lamp for two hours and obtained a browning and finally a blackening of the epidermis of the exposed tissue They estimated that death of the epidermal cells occurred after 15 minutes' exposure to the lamp because of the failure of these cells to react to plasmolysing solutions. Stoklasa (10) repeated these experi-

ments and obtained a similar result. Other investigators have found plant tissues injured by intense light. A great many writers have discussed the effect of ultra-violet light on cellular life in the sterilization of water.

The mercury vapor lamp is presumably the most successful lamp for the ordinary generation of light nearest approaching solar radiation in quality. As no equipment of this kind was available, an arc light of simple construction was employed in the following experiments. A wooden frame was made to hold two $\frac{5}{8}$ inch cored carbons and permit their temporary adjustment in any fixed position. The ordinary 110 volt alternating electric current was used as a source of energy. The strength of the current was lessened by a rheostat composed only of two pieces of common iron baling wire, each 50 feet long, wound into spiral form. The carbons were boiled for 1.5 hours in a concentrated solution of ammonium-molybdate to increase the quantity of ultra-violet light omitted. With fairly constant attention this apparatus gave excellent service, producing a steady light of considerable intensity. The quality of the light is not known, though Schunck (9) says carbons treated as these were emit an abundance of light as short as λ 2,300. In using the light the eyes were always protected by dark glasses. Positive sunburn to the face and arms resulted from an exposure of 0.5 hour at the ordinary working distance.

The bean pods were exposed to the arc light when they had reached a point in their development fairly free from chlorophyll. On a few the peduncle end of the pod showed a green color. This is the stage of development at which the pods show scalding in the field. The pods were exposed to the light at a distance of 25 cm. for periods of 0.5 and 1.0 hours. It was necessary to choose a time when the greenhouse was cool and the ventilation good, in order to maintain a suitable low temperature in the immediate neighborhood of the pods. The arc generates a great amount of heat. On such occasions the temperature rose to only 25° C. at the surface of the pods, as indicated by thermometers exposed near to the pods.

A change in the appearance of the pods was observed 24 hours after exposure, being fainter at first on the pods exposed for only 0.5 hour. A faint browning and streaking appeared, which gradually increased in intensity during the following 72 hours. It was identical in every respect with the normal sunscald, as previously described (5). Bean plants with ripening pods exposed to the full sun for 5 hours each on March 30 and 31 developed mild sunscald. The lesions on the artificially and naturally scalded plants were identical in every respect.

Microscopic examination showed the epidermal cells to be affected. They had become filled with a brown pigmented substance but appeared unharmed in other respects. On the thoroughly whitened pods the stomatal guard cells, which now contained no green matter, were also affected. The cells did not plasmolyse when treated with strong or sugar solutions.

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The burning of plant cells by chemically active light rays, or light of short wave length, has not been much observed in nature. A thick atmosphere, especially when holding a large amount of suspended

water vapor, is so absorptive of light of very short wave length as to practically prevent its reaching the earth. That some light of short wave length does penetrate to the lowest levels is attested to by the fact that in the tropics cataract of the eye due to coagulation of the epidermal layers of eye tissue is frequently encountered among elderly people (4), although certain conditions of health may predispose the eyes to cataract. In this case, however, the effective wave length is apparently above λ 2,650.

The Colorado bean section, at an altitude of 4,700 feet (1,430 meters) and with a relative humidity of less than 20 per cent for a few hours during the heat of the day, would receive a much larger portion of ultra-violet light than those sections at an altitude varying between sea level and 800 feet (244 meters) and with a relative humidity of from 60 to 80 per cent. Brandenburg (3, p. 862) says that the prevailing lack of moisture in Colorado air is favorable to increased intensity of the direct rays of the sun. Solar temperatures obtained from a black-bulb thermometer in vacuo, exposed to the direct rays of the sun at midday, average during the different seasons about 60° F. higher than the air temperature.

The difference in altitude between western and eastern bean-growing districts may make the sunscald definitely apparent only in the West.

The physiology of the action of ultra-violet light on individual plant cells seems not to be thoroughly understood. Maquenne and Demoussy (8) endeavored to determine this point by comparing the phenomenon shown in leaves dipped in water at different high temperatures, in alcohol, ether, and chloroform vapors, and injured mechanically, with that in leaves sprayed with ultra-violet light. They concluded that the brown color is caused by the action of diastase within the cell acting on the starches which have been released upon the death of the cell. Agulhon (1) finds that ultra-violet light destroys not only the cells but also the active products of the cell. The visible light is active on diastase in the presence of oxygen; the ultra-violet light is able to decompose water, form hydrogen peroxid, and then, in the presence of either free oxygen or only hydrogen peroxid, destroy the diastases by the phenomena of oxidization. Stoklasa (10) says that the browning of leaves exposed to ultra-violet light appears after the protoplasm of the epidermal cells is killed by the chemical action of light. Bovie (2) subjected egg white and ox serum to ultra-violet light in a quartz tube. A coagulum was formed which was similar in all respects to a coagulum formed by heat in these same proteins.

CONCLUSIONS

The sunscald of bean pods is caused by light of short wave length. It kills the epidermal cells of the pods.

The leaves of bean plants may be scalded, resulting in indeterminate symptoms similar in some respects to mild mosaic.

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